St. Bartholomew's Hospital Journal.

VOL. X.—No. 5.] FEBRUARY, 1903. [PRICE SIXPENCE.

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St. Bartholomew's Hospital Journal,

FEBRUARY, 1903.

"Æquam memento rebus in arduis
Servare mentem."—*Horace,* Book ii, Ode iii.

Editorials.

WE have received the following letter:

To the Editor of the St. Bartholomew's Hospital Journal.

DEAR SIR,—As an old student of Bart.'s I am frequently asked my opinion as to what I think of the suggested appeal for charitable contributions to so rich a Hospital. The *pros* and *cons* of the subject have been discussed in the press frequently with a considerable amount of bias, and I find, on the information I have been able to glean, that it is very difficult to give to outsiders an opinion that I am able to back up by an appeal to the facts. I would therefore suggest that, as soon as practical, you should give in the JOURNAL some data whereby we may answer the rather unfriendly criticisms that we frequently have to listen to without being able to answer. A more definite knowledge will lead to more definite opinion.—Faithfully yours,

WILFRED N. SODEN.

We print this as one among many of the same tenor. We think it highly advisable in the interests of the Hospital and of the School to put some of the facts of the case before our readers.

* * *

The gross income of the Hospital is, we believe, about £90,000, but this is nearly all derived from real estate, and the necessary outgoings for management and repairs reduce the net income to about £73,000. This would be, and has been, enough to cover the ordinary expenditure of the Hospital. Indeed, the Governors had been able to lay by a thousand or two yearly for the last few years with a view to the purchase of some more land from Christ's Hospital. But it is evident that it can do very little more. This can easily be seen if the general balance-sheets of several hospitals be compared.

Thus in Burdett's *Hospitals and Charities*—our copy is for the year 1899—

The London, with	770 beds,	spends	£72,630.
St. Thomas's, with	570 ,,	,,	£48,977.
St. George's, with	351 ,,	,,	£36,800.
St. Bartholomew's, with	674 ,,	,,	£63,549.

According to the figures given by the Treasurer in the *Times* of January 16th the average net income of the Hospital is £72,315, and the average expenditure on the Hospital and Convalescent Home £70,933. These figures allow of some annual saving, which saving has resulted—we speak without authority—in a sum of about £30,000.

* * *

Then came the purchase of the additional acre and a half urgently needed for new casualty, out-patient, and special departments, new pathological institute, new isolation block, new college, and new nurses' home. In our present buildings no one can say that patients can be treated or students taught as becomes a great hospital and a great school.

Into the details of the transactions we need not now enter. They may be found in the minutes of the House of Lords Committee which sat on the question of compulsory arbitration. In the end we had to pay a sum which, including law expenses, reached about £250,000 for the acre and a half.

* * *

To meet this demand the Hospital had to sell stock to the extent of £133,000 and to borrow £120,000, transactions which reduce the annual income by £8972, and result in an annual deficit. Let it be remembered that the Hospital have raised a quarter of a million for an extension that all own to be absolutely necessary, without asking the public for a single penny. These figures, as we said, are public property, as they are quoted from the Treasurer's letter to the *Times*.

* * *

But besides this sum paid for land we should think that at least £300,000 will be needed to put up the buildings required.

It is quite obvious that no hospital can ever pay for large capital outlays out of income. It must be content to spend its income to the best possible advantage of the public, and must look to the public once in a hundred years or so to give it a large sum for the renewal of buildings. In our case it is 150 years since we appealed for the money with which the present wards and administration block were built. That money at all events was well laid out.

But it unfortunately happened that our appeal interfered with the regular quinquennial appeal of the London, and the chairman of the London, who thinks all is fair in love, war, and charity, laid his plans accordingly. A regular attack was organised, and the batteries were opened as soon as the Lord Mayor announced his readiness to head an appeal.

* * *

The line of attack was as follows. We were said to be an enormously wealthy body, with an income £25,000 in excess of expenditure, placed on a site worth £1,750,000, in a position of little use to the public, and of an unhealthy and undesirable nature. We were from merely sentimental considerations holding to the present site, and retaining the present germ-sodden and antiquated buildings, while all the time we had a glorious site of fifteen acres in St. Luke's which was our own property, to be had for a very small compensation to be paid to the existing tenants. If we behaved like honest men we should sell our present site, and with the proceeds not only buy the other site, but build a hospital for 1000 beds, and put money in our pockets too. And yet we had the impertinence to come to the citizens of London and ask for money for our wicked and preposterous scheme, while all the while the great London Hospital itself was starving. This, we think, fairly represents the case for the prosecution as set forth in the public press.

* * *

It may at once be stated that every one of those statements was incorrect.

1. As above mentioned, our net income was only just above, and is now below our expenditure.

2. The value put upon our present site by the advocates of the policy of removal varies between £1,000,000 and £1,750,000. The higher of these estimates rests upon no basis of authentic figures, but is apparently evolved out of the inner consciousness of the imaginative editor of the *Hospital* newspaper. The lower would seem to rest upon the rate we had to pay for the land recently acquired, and it is the opinion of eminent surveyors that that price was excessive. It is more probable that the actual value is much more accurately estimated at £750,000.

3. It is the only great hospital for the City itself and for the great industries which lie north of the river, and it is in about the most accessible spot in the whole of London.

4. The site is a picked one. It is on high ground, on a gravel soil, with a large open space of about six acres to the north of it.

5. The buildings leave many things to be desired, but they are a good deal better than the wards of some other hospitals, and our results are at least as good. The staff actually sent a memorandum to the Treasurer, which he published in the *Times*, that the Hospital was one of the healthiest in London.

6. There is no available site of fifteen acres in St. Luke's at all. The Hospital is the ground landlord of about eight acres, of which three acres is held on a perpetual lease, the remainder on leases with from twenty to thirty years to run, intersected with streets, on a damp clay soil. To obtain its use St. Luke's Hospital would have to be bought out, the other leaseholders and several incutting owners compensated, and Parliamentary powers sought to close the streets. Considering the value of town property and the cost of legal processes, even an amateur in such matters may be so bold as to estimate that a sum of over a million would be required to build an adequate hospital on such a site, for which we should only have the £750,000 obtainable for the present site and a little nest egg of at least £250,000 of debts.

* * *

These are all plain and simple facts, which anyone can find out for himself. Most of them are published, the remainder have been publicly stated. But editors, with the exception of the editor of this JOURNAL, are not over-fond of accuracy. Truth is apt to be dull, and requires some little trouble to discover. Fiction—the wilder the better—is the paying thing; an attack—the more personal the better—the most relished. If you cannot tell a lie about a public man, tell several about an ancient and an honourable institution,

and verily you shall have your reward, for the paper will sell. We may piously hope that in the next world there is for such editors a small but warm corner reserved between Ananias and the naughty little boys who mocked Elisha.

* * *

IN the hurry consequent upon our efforts to bring the publication of the JOURNAL up to time the correction of the proofs of the last number was very imperfectly done, a fact which accounts for the following list of errata in Sir William Church's article. For these mistakes we offer our readers and the distinguished writer of the article our sincerest apologies.

Page 50, *for* Podalisius *read* Podalirius.
" 51, " hurriedly " learnedly.
" 51, " Autyllus " Antyllus.
" 52, " Columbus " calculus.
" 52, " Herodotus " Herodicus.
" 53, " Rhayes " Rhazes.
" 53, " Salcormen " Salernum.
" 54, " suction " coction.
" 55, " these " those.
" 56, " career " careers.

* * *

BY the election of Dr. A. E. Garrod to fill the vacancy created by the retirement of Sir William Church, and of Mr. McAdam Eccles to the Surgical side, our staff is once more complete. To the present generation of Bart.'s men Dr. Garrod's appointment will be especially welcome, coming as it does after so many years' faithful service to the Hospital in the various junior offices, whose duties he has so admirably performed.

* * *

LAY criticism is often frank and always instructive. The other night a late member of the Junior Staff was driving in a hansom to an address in Wimpole Street, when he was suddenly startled by an inquiry through the hole in the top as to whether he was acquainted with a certain well-known London surgeon. On answering in the affirmative, and asking why the cabman wanted to know, he was told, "Well, 'e ain't no bloomin' good at 'ernia, 'e ain't. 'E did what 'e called a radical cure on me, 'e did, and 'e ain't no *bloomin'* good at 'ernia."

"Neurasthenia:" a Critical Inquiry.

By THOMAS J. HORDER, M.D.

(*Read before the Abernethian Society, February 5th*, 1903.)

MR. PRESIDENT AND GENTLEMEN,—So much confusion has gathered around the term neurasthenia that I need offer no excuse in bringing the subject before the Abernethian Society this evening. I am not so sanguine as to think I can aid much in an inquiry concerning the matter, but I shall at least hope that I may not end by leaving the confusion worse confounded.

"There are three stages of evolution through which all new ideas, before they are received into the full and unchallenged fellowship, have passed, are now passing, or are destined to pass. First, the stage of indifference; secondly, the stage of denial; thirdly, the stage of contests of priority."

This is a paragraph from the second edition of Beard's *Practical Treatise on Nervous Exhaustion, or Neurasthenia*, published in New York in 1880. We will allow that the first two stages in the history of a new idea are often as Beard describes; but the third stage is, or should be, the stage of careful criticism—a necessary and valuable stage, the importance of which is much greater than a contest as to the proper distribution of the laurels on a matter of discovery. This fourth stage we are content to dismiss by accepting Beard's postulate that "the first honour in science belongs to him who organises." The previous stage, however, that of careful examination, we prefer to have "long drawn out," knowing that the idea will not suffer from this treatment if it be worth ultimate acceptance. It is to this inquiry that I wish to contribute this evening.

Let me remind you briefly of the state of opinion on this subject previous to Beard's efforts at organisation. Nervous diseases were conceived of as falling into the two groups *organic* and *functional*, a distinction we still hold, but hold loosely, prepared to see, from time to time, examples of the second group move into the first as our methods of pathology advance. This transference has always been regarded as satisfactory and to be aimed at; for the more closely changes in function were studied, the more surely was it found that they corresponded with changes in structure. In all other regions of science than in medicine force and matter had long since been admitted to own inseparable relationships, changes in the one going hand in hand with changes in the other. So that to premise that change in function meant change in structure, however slight and difficult to demonstrate, seemed a safe proposition to work upon. As changes in structure are of more certain recognition, and serve as safer criteria of disease, the organic basis of disordered function came to be the ultimate object of our search. These efforts have been rewarded by the discovery of the pathological processes underlying many nervous diseases previously called functional.

But amongst the various morbid functional conditions still remaining it was found possible to separate off certain groups which were sufficiently recognisable as definite entities. Some of these were sharply limited, as epilepsy and chorea; but others graded more or less insensibly into each other, into the remaining mass of nervous symptoms, and into health itself. These others included hysteria, hypochondriasis, and the forms of insanity. The residue, a

heterogeneous mass of symptoms, went by the name of "nervous debility," "nervous exhaustion," etc.

This was the state of things in 1868, when Beard came with his attempts at "organisation" of this residue of symptoms, and of them formulated a disease to which he gave the name "neurasthenia." Beard's doctrine has gained acceptance in most countries and in the minds of many skilled and thoughtful men; so that to-day most of the text-books of medicine contain an account of neurasthenia, clinical lectures are given on the disease, and "cases" of it demonstrated. Here in England it is a common diagnosis at Queen-Square, the home of our best teaching in neurology. Allbutt, in his *System of Medicine*, refers to Beard as at last being "avenged," as though a man could suffer wrong by promulgating a doctrine which was coldly received by his fellows, or find his wrong redressed when they ultimately entertained it.

Beard gave us no definition of neurasthenia, but made a full list of the symptoms suffice as an expression of it. Some of Beard's followers have attempted to reduce their conception of the disease to a definition, but there is very little actual agreement between them. Von Ziemssen considers neurasthenia to be "a state of functional debility of the nervous system manifesting itself by defects in the psychical, reflex and inhibitory centres, and also in the conducting paths." This is, therefore, no less than saying that neurasthenia is a state of lowered functioning of the whole nervous system. Savill, a recent English writer on the subject, understands by his use of the term "an irritable weakness of the entire nervous system, characterised (when the brain is chiefly affected) by hyper-sensitiveness of the sensorium, by headache, inaptitude for mental work, disturbed sleep, and irritability of temper; and (when the spinal cord is chiefly affected) by general weakness, restlessness, nervousness, and vague pains; and usually accompanied (in both forms) by various phenomena referable to the vaso-motor and sympathetic systems."

As regards the essential nature of the disease, Beard's theory was that the various symptoms are due to local hyperæmia of the particular organ through which the symptom manifested itself, this hyperæmia depending upon a disturbance of the nervous system by which waste and repair are ill-adjusted, the disturbance being set up by various causes, but especially "over-civilisation." Arndt considered the primary factor to be defective development of the nervous system, and possibly also of the blood. Most authors attribute the disease to a combination of hereditary predisposition and nerve strain.

To Beard's description of the symptoms of neurasthenia little of importance has since been added. It is, however, usual to consider these symptoms under certain heads, and I intend enumerating them in this way,—without any description, however, as they are self-explanatory.

1. *Cerebral symptoms.*—Deficient mental control; mental irritability; mental depression; defective attention; lack of memory; hopelessness; morbid fears, to which a whole string of Greek names is given, as astraphobia, agoraphobia, claustrophobia, monophobia, etc., up to pantaphobia, or fear of everything; fidgetiness; insomnia; bad dreams; night terrors; drowsiness; pain, pressure, and heaviness in the head; hemianopia; asthenopia, with contracted field of vision; deafness; *muscæ volitantes;* noises in the ears; giddiness; atonic voice; dilated pupils; tenderness of the scalp.

2. *Gastro-intestinal symptoms.*—Tenderness of teeth and gums; dyspepsia; deficient thirst and incapacity for assimilating fluids; hæmatemesis.

3. *Cardio-vascular symptoms.*—Palpitation; tremulous and variable pulse; tachycardia; cold hands and feet.

4. *Spinal and peripheral nerve symptoms.*—Tenderness of spine; general hyperæsthesia; local anæsthesia; sacralgia; shooting pains; cramps; temporary paralyses; tremors; convulsive movements, especially on going to sleep; pruritus.

5. *Sexual symptoms.*—Spermatorrhœa; partial or complete impotence; irritability of prostatic urethra.

6. *Miscellaneous.*—Abnormalities of the secretions; hyperidrosis; dysphagia; rapid decay and irregularity of teeth; restlessness; ticklishness; profound exhaustion or sense of fatigue, unaccompanied by positive pain; gaping and yawning; sensitiveness to the weather, and to changes in it; oxaluria; phosphaturia.

The multitude and the great range of these symptoms might well justify physicians in hesitating to adopt Beard's idea that they should be summed up as manifestations of a particular disease. This difficulty is admitted by Beard and his followers, but does not deter them from considering the symptoms as correlated. Allbutt warns us that neurasthenia is *not* a "hotchpot, into which odds and ends of unconsidered trifles are to be thrust away for lack of definite affinities." It is these "definite affinities," however, that we must have shown us before we concede the main point at issue. Of the actual existence of the symptoms just enumerated there is no doubt; all of them may be met with frequently by any careful observer or, we feel bound to add, careful *listener* For it will be noticed that they are for the greater part purely subjective; the objective signs of neurasthenia are confessedly few, or are wholly absent. The real question is whether these symptoms are sufficiently correlated to constitute an entity worthy the name of a specific disease. Incidentally the question might be asked whether we are ever right in building up a disease in the absence of objective signs.

A further issue is whether, if nothing be gained by changing the old notion of "nervous debility," which connoted a variety of symptoms not necessarily connected, for the new one of neurasthenia, something is not lost by a too premature stringing together of symptoms that are

better kept isolated, awaiting further pathological research. It is not an uncommon thing, as some one has pointed out, to deceive ourselves into thinking that we have advanced a step when we change a Latin name for a Greek one. But it may be worse than that: we may thereby be induced to mark time when we should be marching. I shall hope to illustrate this objection to the notion of neurasthenia later on. Meanwhile let me point out that it is to be expected, from the very complexity of its structure and from the universality of its distribution, that disorders of the nervous system will longest resist our attempts at segregation.

I will now proceed to deal with some of the symptom groups included under the notion of neurasthenia. To begin with the cerebral group, I am of opinion that the term neurasthenia is used to cover many symptoms where the real defect is a mental one. It may be that we have become too polite to ascribe our patients' symptoms to mental weakness, or that we have narrowed our conception of mental defects to the grosser kinds of insanity. It is certain that there is a great tendency to sink the mental factor in our consideration of the pathogeny of nervous disorders. Many conditions to-day adorned by the grandiloquent title "neurasthenia" would have received much shorter, and I think truer shrift from our fathers. There are many degrees of weak-mindedness that fall short of actual lunacy, but it is still important to remember that they are allied to it rather than to nervous diseases proper.

The various *obsessions* or imperative ideas, upon which all writers on neurasthenia lay considerable stress, are of this nature. When "an idea or group of ideas invades the mind automatically to the exclusion of others," it argues mental weakness. These obsessions are of two kinds, morbid fears—of places, open or closed, high or low, of diseases, contaminations, animals, lightning, etc.; and morbid doubts—varying between that distrust of self which acts but as an undesirable handicap to life, up to the most absurd and prolonged indecision,—say as to which boot a man should take off first on going to bed. Obsessions are sometimes found in otherwise healthy persons—probably we all know of friends who possess them,—and this is a notable instance of our inability to mark off the sound mind from the unsound by any rigid line. But where these imperative ideas are marked, and interfere with a person's proper relations to his customary environment, his condition comes within the realm of insanity.

But a second and much larger group of symptoms attributed to neurasthenia should, I think, be classed as signs of *Hypochondriasis*. I define hypochondriasis as a disease of the mind leading to an excessive central representation of various organic sensations. In the broadest sense, then, this also is a form of insanity, but like the obsessions it forms a border-line condition. I do not hold with those who say that, owing to growth of knowledge, hypochondriasis is becoming less common. I think it a very common disease indeed, and though the "neurasthenia" school claim that they carefully mark off their cases from cases of hypochondriasis, I should myself unhesitatingly give many of their recorded cases this latter diagnosis. They mark them off only from the more obvious cases of hypochondriasis, handing these last over to the alienist, as if (to use Dr. Mott's words) "to bribe him not to claim the whole." Arndt says that hypochondriasis is a cardinal symptom of neurasthenia, and thus admits the mental element in the cases; but I consider that in not a few of the cases the mental element is the chief one.

In cases of hypochondriasis a family history of insanity is very common, and if not of insanity, then of some other neurosis, such as epilepsy, alcoholism, chorea, or asthma. With asthma it seems to have a special association. I have at present under treatment a young man, aged thirty, who suffered from asthma between the ages of seventeen and twenty-five, and still occasionally has attacks. His family history includes two important points: an aunt had spent some months in a private asylum, and the patient himself was the youngest of three children born after his father's second marriage, which took place when the widower was sixty years old. He complains of insomnia, mental depression, and weakness of one leg that comes on after very little exertion; it is thinking about this leg that keeps him awake at night. There is nothing amiss to be discovered after repeated examination, and this fact, together with the combination of neurotic inheritance and asthma, makes the diagnosis of hypochondriasis tolerably certain. The immediate cause of the trouble seems to have been undue mental concentration upon a matter involving the affections, in which the element of indecision entered largely. This, by the way, is no infrequent cause of mental break-downs. Many of the cases that go labelled "overwork" go labelled wrongly; that is if, as is usual, the meaning of "overwork" is overwork of the intellect merely. It is not enough to know how much brain-work a patient is doing; more important than this is the knowledge of how much worry there is connected with the affections, the ambitions, the making both ends meet or overlap, as the case may be. And let me incidentally refer to the great difficulty there often is, not only in determining this present mental element in a patient's case, but also in getting accurate information as to the past mental history of the family. This is of the utmost importance in all functional nervous affections; and from the delicate nature of the questions to be asked it is often advisable and justifiable to get to know some intimate friend of the family, or some sensible member of it other than the patient, to whom to confide the importance of the inquiries.

The commonest complaints of the hypochondriac refer to the stomach and bowels, the heart, and the sexual organs. But any organ may receive the patient's dis-

ordered attention, and in some of the cases the very number of the organs complained of is itself a valuable means of deciding the nature of the case. The physical sign of hypochondriasis, if I may put it so, is an hour's discourse from the patient upon his various symptoms, or a written account of them extending to several pages. I had one patient who brought me a diary containing a record of his observations during a whole week, beginning early each morning with variations in the reddish deposit of urates he noticed separating from his urine, and ending with the difficulty he felt in keeping his eyelids open late at night.

The essence of this disease seems to be that the thousand and one transient organic sensations which never rise up into consciousness in the healthy mind—or rise only to be summarily dismissed—attain in the hypochondriac important dimensions, and claim his constant attention. The seat of the disease is in the brain, though it is interesting to note that this doctrine has only been universally admitted during the past sixty years. The defect being chiefly mental, the treatment must be largely mental also. But in some cases excellent help may be derived from the use of cannabis indica, taking care to select a specimen of the extract that is active. This drug seems to act by lowering the central representativeness of many peripheral stimuli, and thus producing a state of *bien-être* by freeing the patient from the tyranny of his own sensations.

The whole group of symptoms dignified by the name of sexual neurasthenia we may transfer to the less fashionable category of hypochondriasis. It was here that Paget put them, and there is no reason for thinking they are other than manifestations of mental weakness evoked by organs which are specially prone to arouse morbid interest.

Of the cases of hypochondriasis where there is recognisable visceral disorder underlying the mental condition, the women possessed of movable kidneys are conspicuous. Doubtless some of these suffer before they know of the defect inside their abdomen, though their symptoms are often only of a mild dyspeptic character; but their sufferings are doubled when the knowledge comes, and this experience makes it highly undesirable that doctors should tell these patients what they find on examination. A somewhat amusing incident happened to me at the Great Northern Hospital some three years ago. A cook, tall and thin and sallow-faced, brought the housemaid with her saying that she knew her colleague had a floating kidney, because she was suffering in exactly the same way as she herself suffered before she was operated upon for a floating kidney of her own. I examined the housemaid for a movable kidney, but with a negative result; but on examining the cook I found that just beneath a very lax linear scar in the right side of the abdomen my hand came down upon the kidney, easily movable, and capable of being pushed almost across the lumbar spine. It required considerable forbearance to hide from her the real state of her organ. It must always be borne in mind that a movable kidney may be an incidental event in a case, the symptoms of which may be quite independent of its existence. Of course, as in the cook, the symptoms may be relieved by a nephrorraphy which fails to secure the organ; but they may also be relieved sometimes by the actual cautery.

It is doubtful if there is any real difference between hypochondriasis and *hysteria* apart from the sex, age, and temperament of the individual affected. Hysteria, we are all agreed, is a definite disease. The underlying cause seems to be the operation upon the bodily functions of a subconscious state. Here again there is a morbid degree of representativeness, not remaining in the cerebrum as such, however, but reacting upon the body in the production of the various motor, sensory, and sympathetic states so well recognised. Given these particular stigmata, the diagnosis of hysteria is well grounded and sufficient, and to change the name for neurasthenia by no means adds to our scanty knowledge of the condition.

The *cardio-vascular* symptoms included under this notion of neurasthenia form a very common clinical picture, and a very important one. A slight degree of anginal pain, various sensations such as palpitation, throbbing, fluttering, etc., undoubtedly referable to the heart, a frequent or infrequent pulse, and dyspnœa,—when we are faced with this series of symptoms the question of some affection of the myocardium is very difficult to settle. The subject has been dealt with fully by Dr. Gee in a clinical lecture published in this JOURNAL recently, under the term "Weak Hearts." The pathology of the disease, whether primarily a muscular or a nervous defect, probably differs in different cases, according to the sex of the patient and the causes at work; it is perhaps both nervous and muscular in many cases. Most of the cases seem to run a disappointing course, especially those beginning in persons over fifty years old.

I think that when we can put our finger upon some particular organ whose functions are disordered, and the disorder shows itself in various nervous symptoms, we do well to speak of the condition as a disease of the particular organ rather than of the nervous system. It is obvious that disease must chiefly manifest itself to us through the nervous system, and these cases I have referred to are best spoken of in terms of the heart, although they do not show any gross objective signs.

Of the *gastro-intestinal* symptoms I might say the same thing. It is needless to argue that many cases of dyspepsia own a primary nervous origin; this we were prepared for when we discovered how dependent the digestive secretions, movements—indeed, the whole mechanism of digestion,—are upon nervous influences. And recent work in this direction shows that we must still further enlarge our conception of this element in digestion. We may admit that nerve defects are the primary cause of a particular case of

dyspepsia, and still regard it as a case of disordered digestion.

If my contention be allowed, many cases of neurasthenia are better classified elsewhere. But there is another danger in conceiving of neurasthenia as a definite entity: we may rest too content with this as a diagnosis, and forget that the beginnings of many diseases may comprise similar symptoms. These diseases must always be borne in mind whenever the picture of "neurasthenia" arises. They include particularly Graves' disease, myxœdema, arterio-sclerosis, aneurysm, myocardial degeneration, Pott's disease, hepatic and renal calculus, new growth of the stomach, kidney, or other viscus, and dilated stomach. These are diseases where the lesion is not primarily nervous. Of nerve diseases proper we must especially consider tabes dorsalis, insular sclerosis, and cerebral tumour. If either of these organic diseases be really present, the word neurasthenia, used to cover the nervous symptoms, is really more a nuisance than a help.

Turning now to the cases of functional nerve disease which cannot be placed in either of these categories, we are struck by the frequency with which they follow, or accompany, very definite *causes*. And this being so, it seems unadvisable to group the cases together as forming a distinct disease. I might add, the very fact that the causes admitted by writers on neurasthenia are so many and so different is presumptive evidence against the view that we are dealing with a specific form of nerve disease.

(1) *Trauma* produces the worst and the most marked cases. The conditions formerly spoken of as "nerve shock," "spinal concussion," "spinal irritation," and so forth, are being talked of as "traumatic neurasthenia." And these cases are often pointed out to us as yielding convincing evidence of the uniformity and consistency of the disease.* I must confess I have difficulty in following the argument. To allow that these cases fall into a definite group with a well-marked symptomatology does not militate against the claim that similar symptoms following other and very different causes should receive different consideration. Analogies are always treacherous in medicine, but one is tempted to say that, on this argument, coma should be treated of as a definite disease because its symptoms are more or less similar whenever it arises, whether it be due to uræmia, epilepsy, or concussion of the brain. These traumatic cases own a clear cause and a simple one, and it does not seem too much to assume that the process of their production is very different from that in many other cases of nervous debility due to other causes. Here, for instance, the symptoms might be readily explained upon mechanical grounds, either to actual disorganisation of, or to minute hæmorrhages into, nerve substance, or to a disturbance of the delicate central nerve connections interfering with nutrition, and too fine for demonstration.

So far as I am aware there is every transition between the traumatic cases termed "concussion of the brain," with instant and grave impairment of cerebral function leading to coma, and cases labelled acute traumatic neurasthenia. Yet in severe cases of cerebral concussion structural changes may often fail of demonstration.

(2) *Sunstroke* is the cause of a set of symptoms as deserving of the name neurasthenia as any other combination. Yet it seems highly improbable that the actual underlying defect in the nerve tissues is the same as in, say, trauma.

(3) We are most of us familiar with the havoc *influenza* can play with the nervous system, central and peripheral. Other acute specific fevers, such as *typhoid*, lead to functional nervous affections, but none so often as influenza. The increase in so-called neurasthenic disease of late is doubtless largely due to this cause. There is scarcely any disorder of the nervous system which may not be a sequel of influenza. I recently treated a lady aged sixty-six for a condition clinically indistinguishable from paralysis agitans; there was, in addition, marked loss of memory for recent events. The nervous symptoms came on within three weeks of the fever. As one would suppose, the course of the illness was very much more favourable than true progressive paralysis agitans. To classify these various influenzal conditions in the same category as others due to widely different causes seems highly undesirable. It suffices to designate them "nervous debility following influenza." That they are toxic in causation seems fairly certain.

(4) The *climacteric* is occasionally accompanied by symptoms included under the name neurasthenia. When we have to deal with such a case, we may hold out a fair prospect of recovery after the menopause is over, provided no signs of organic disease are discoverable, and always assuming there is no previous or family history of insanity. Here again, ignorant though we are of the nature of the changes going on in a woman's nervous system at this epoch, it is scarcely likely that they have much affinity with the changes due to mechanical shock or the poison of an acute infection.

(5) But I might continue to deal with the causes of neurasthenia in this way at great length. The causes, like the cases, are very numerous and very dissimilar. Twelve months ago I treated a patient for a distressing form of nerve weakness, which I thought I had sufficient evidence for ascribing to *tobacco*, and a resulting chronic gastritis. Some months before this the patient had had a cardiac attack. with sudden pain and palpitation, which seized him as he was telling an after-dinner tale. He was at this time smoking about six ounces of tobacco a week, with one or two cigars and cigarettes that went uncounted. He was a powerfully built man, aged 30, a keen sportsman and an excellent shot, and would, I suppose, be one of the neurasthenic

* Dr. Allbutt, in his *System*, says, "On consideration of the traumatic cases the reader, if he were inclined to regard neurasthenia as an indefinite medley of inconstant symptoms, may be converted."

patients spoken of by Clifford Allbutt and others as being robust and in seeming good general health. He was constipated, his tongue was coated with a dark brown fur, into which I could sink a pencil of ordinary thickness, and he complained of a burning sensation in the throat and beneath the sternum. Apart from these things, however, he had no signs of disease. But he was driven almost crazy by an aggravating irritability of temper, of quite recent date, which compelled him to lock himself in his room lest he should vent it upon his inoffensive wife. The office boy, I gathered, was not allowed the intervention of a locked door, so suffered accordingly. This restless irritability might come on at any time, and any one handy might know of it. The thing preyed upon his mind, and the result was insomnia, inability to work (he was an architect), and a most dreary state of hypochondriasis, resulting in long consultations and letters in which various strange sensations were described up and down his body. He came to me one morning, without appointment, saying that when he left home after breakfast he had no notion of calling, but that "an attack" had come on, and he scarcely felt answerable for what he might do. This patient improved rapidly when his tobacco was cut down, and cannabis indica seemed to benefit him considerably. He is now, I believe, quite well.

But there are those who admit that the term neurasthenia is being wrongly used, cases being included under it which should be placed elsewhere, or in our present state of knowledge placed nowhere, yet who urge that there is a class of case, the symptoms of which possess sufficient definite affinities to form a series, and deserving this special name. Such a position is, of course, a reasonable one to adopt. But we still lack any clear statement as to these cases, and of the criteria by which they are recognised. We read the expression "neurasthenic patient" too often to avoid the inference that it means the same thing as "nervous patient," and a nervous patient may have any disease or none. Now it is confessed that there are no recognisable structural changes in the disease; there also seems to be no criterion of its symptoms; the cause is admittedly not one, but divers. Are we not correct in concluding on these grounds that the disease itself is "not proven"?

When we have separated off the cases having recognisable mental defects, those attributable to clear, well-understood causes, and those better dealt with under diseases of the particular organs through which the symptoms arise, we have remaining an indiscriminate set which it suffices to include under the term "nervous debility." Or the term "neurasthenia" would serve did its use not carry also the notion of a definite series constituting a disease. It seems much more consistent with our mode of procedure in other regions of medicine to wait for pathological relationships rather than to build up these varied symptoms into a single disease.

Fresh lines of research are being indicated in several directions. The presence of toxic products of abnormal nerve katabolism in the circulation, such as the cholin group; the chromatolytic and other changes going on in nerve-cells; the comparative study of nerve-cells under various conditions in insects,—these are three pathological methods still in their infancy. So that clinicians need not hurry to force cases into ill-fitting classes. If they do, it is fairly certain that the pathologist will insist upon breaking these up again.

Turning for a moment to the *course* of neurasthenia, and to the effects of *treatment*, these also seem to argue against the uniformity of the condition. Knowing how diverse the causes are, we should expect successful treatment to be equally diverse, and such is the case. Beard admitted that no general lines of treatment could be laid down; the cases must be treated individually. Beard's followers, however, are often less wise, and Weir-Mitchell treatment has become for some of them the Morrison's pill for all cases. But there are cases of nervous debility that get manifestly worse under this treatment, that is if the three elements making it up are strictly carried out.

On the other hand, we must not be led astray by the fact that the results of Weir-Mitchell treatment are often very good in many of the cases,—led into believing, I mean, that this proves them to be of the same nature. I think, by the way, the reason it benefits a large number is the reason why it benefits cases of hysteria,—it deals with the mental factor; which notion supports the view I took just now that many cases of so-called neurasthenia are in reality cases of hypochondriasis. But I suppose there are very few people—probably there is none of us here, met together on an evening three parts of the way through a long winter session—who would not benefit considerably by six weeks' Weir-Mitchell treatment. If it comes to that, judging by the dismal pictures we sometimes see drawn of our nervous state generally, it seems likely that the whole civilised world would vastly benefit if it could be clapped into a huge Weir-Mitchell institute for a month. The question that naturally arises is, Who would bear the expense? which is the question many a patient asks when his doctor prescribes this particular treatment.

To touch upon another and broader aspect of the question: is not the line of health being drawn rather too fine? Health, as we all know, is no fixed state, but is inclusive of certain oscillations, which must be regarded as variations rather than disturbances of the normal. And if in one individual his health is a different thing at different times, much more impossible is it to compare the health of one individual with that of another. Take the matter of insomnia. Sleeplessness is but a relative term; enough sleep for one man may be sleeplessness for another. There are short-sleeping families and long-sleeping families. It is the same with constipation. "It is no law of nature,"

it has been said, "that the bowels should be relieved punctually once in twenty-four hours." One is sometimes consulted because, do what he will, a patient cannot get an action of his bowels oftener than every second day. If you ask you may find that his father had the same difficulty, and perhaps *his* father also, and none of them probably suffered any ill effects.

These are trite remarks, I know, and I might add to them indefinitely, but the facts underlying them are apt to lose their significance when we deal with the nervous system. Our methods of examination to-day are much finer than they ever were before, and we can more easily detect slight variations from the average in function,—so much so that the "average" is getting lost sight of as a standard, and an ideal of nerve health is unconsciously being used instead.

Some of you may be asking, "Isn't all this a mere juggle with words? What boots it whether a nervous man is called a neurasthenic or whether he is said to suffer from nervous debility?" But in medicine the success of our methods turns a good deal upon our proper use of words. And here is not only a matter of the choice of a word,—as I have said, the mere *word* neurasthenic is as good as any other; the *idea* it conveys, however, I consider an erroneous one, or, I would prefer to say, an unnecessary and dangerous one.

Conclusions.

As a summary to these rather sketchy observations I shall submit the following conclusions:

1. There are many symptoms of functional nerve weakness, both central and peripheral, which do not fall into sufficiently definite series to constitute special diseases. They are at present sufficiently summed up by the name "nervous debility."

2. If the word neurasthenia meant no more than "nervous debility" its use could not be objected to, but its introduction brought also a new conception,—it was supposed to connote a set of correlated symptoms, and thus form a separate entity. Used in this sense the word is objectionable, and for these reasons:

(*a*) A large number of cases are better grouped as mental affections, and especially as hypochondriasis, a common disease, where the fault is an abnormal mental representative of peripheral sensations.

(*b*) Those having symptoms referable to particular organs are best considered as diseases of these organs, even though it be understood that the primary defect is one of innervation.

(*c*) When the symptoms accompany definite organic diseases it suffices to describe them as symptoms of those diseases, and not as a new disease.

(*d*) When the symptoms follow on a clearly traceable cause, *e. g.* influenza, trauma, sunstroke, it answers all the present purposes of Medicine to speak of them as nervous sequels of these conditions.

(*e*) Cases not falling under either of these heads do not show sufficient cohesion to justify their being grouped together as a definite disease.

Discussion on the Treatment of Eclampsia.

Opening Remarks by Herbert Williamson, M.A., M.B., M.R.C.P.

THOSE conditions of the puerperal woman which we class together under the term "eclampsia" are so different in their origin, in their course, and in their remote effects, that it is impossible to consider their treatment in a rational spirit unless we group the cases into certain broad subdivisions. I shall endeavour to show that under this term we include possibly several distinct pathological conditions, but I shall confine my attention chiefly to what I believe to be the two commonest forms—two big groups of cases, which in their pathology and ætiology differ entirely from one another,—which have little in common beyond the fact that in both of them convulsions occur.

What do we mean by eclampsia? Dakin * says, "The convulsions in cases of puerperal eclampsia are of an epileptiform character. They occur before, during, and after labour. They are always associated with albuminuria, and are now pretty universally believed to be due to renal insufficiency, or, at all events, to the retention of some waste products which are nominally excreted by the kidneys."

This definition is unsatisfactory in two respects. (1) Cases of eclampsia are recorded in which there has been no albuminuria. (2) It is by no means even "pretty universally" believed that the convulsions are due to renal insufficiency or to the retention of waste products.

Hermann† says, "The name denotes epileptiform convulsions coming on during pregnancy, labour, or childbed, and depending upon a disease of the kidney which is peculiar to pregnancy."

A little later in the same article, under the heading of Morbid Anatomy, we read, "Very different conditions of the kidney have been found; . . . diseases such as occur apart from pregnancy may be found after death in eclampsia; . . . in some cases the kidneys have shown no signs of disease appreciable by the unaided senses." This is hard to reconcile with his previous definition, nor is his position elucidated by his final summing up: "From these different observations, made by independent patho-

* *Handbook of Midwifery*, p. 509.

† Clifford Allbutt's *System of Medicine*, vol. vii, p. 797.

logists having no hypothesis to support, it follows that although the kidneys often look like inflamed kidneys, yet the lesion of puerperal eclampsia is not nephritis, but an acute degeneration such as is caused by blood-poisoning."

This is perfectly true of one of our two common classes of case. But not only does the kidney show these lesions, every organ in the body shows them—liver, spleen, lungs, heart, and brain. Surely here the kidney lesion is not the cause of the eclampsia or toxæmia, whichever you prefer to call it; it is but one manifestation of a general condition.

At present I know of no satisfactory definition of eclampsia, and it is a very difficult matter to frame one which shall include all those forms of convulsions occurring in pregnant women which we now group under this term.

The pathology of eclampsia forms no part of this discussion to-night. It is still obscure and uncertain; the theories have been many and brilliant, the facts few and hardly gleaned. One generation believes it to be a direct possession of the evil one; their children discuss thyroid inadequacy and embolisms of villi.

We have, however, now seen the dawning of a scientific pathology; but our knowledge is retarded, and our treatment often rendered impotent, by our failure to grasp the fact that eclamptic convulsions may arise from very different causes, and may require very different treatment. So that the first point I would endeavour to establish to-night is, that what may be perfectly good treatment for one case of eclampsia may in another, which has a quite different pathological basis, prove worse than useless; and I would urge that we should endeavour to discover, as far as in us lies, what is the cause of the train of symptoms to which we give this name.

I will ask you to compare and contrast two cases which were admitted into Queen Charlotte's Hospital within a few days of one another at the time when I was resident medical officer there.

The first was a woman who had previously borne four children. In her first two pregnancies she had good health, but in both the third and fourth she suffered from severe vomiting, headache, and œdema of the legs. When admitted into Queen Charlotte's Hospital (under the late Dr. Greg) she was in a semi-conscious condition, with much œdema of the face, legs, and vulva; she had already had a convulsive fit.

After admission she passed very little urine, only four ounces in twelve hours, and this small quantity when boiled became quite solid with albumen. There were numerous casts in the urine—blood, epithelial, and granular. She was quite unable to see, and upon examining the eye grounds marked albuminuric retinitis was found. Very shortly after admission the fits recurred, and it was decided to hasten delivery. After the birth of the child there were no more fits. The patient slowly recovered to some extent, but with hopelessly damaged kidneys, and for many weeks was in this hospital under the care of Dr. Hensley.

Contrast this with the second case.

On November 28th a girl æt. 18 was admitted, in labour with her first baby. She stated that she had been well throughout her pregnancy, had never suffered excessively from vomiting, but that for the last three weeks she had noticed that her legs became slightly swollen towards night.

When I first saw her she was in labour, she complained of headache, and there was slight œdema of the legs, of the vulva, and of the lower part of the abdominal wall. The urine was of sp. gr. 1018, and contained a slight cloud of albumen.

During the second stage of labour, which lasted three hours, she vomited twice; her baby was born at two o'clock. I was not present at the birth, but was called to see her shortly afterwards on account of a ruptured perinæum; this was torn back into the rectum, and at 3.30 she was given chloroform, and I repaired the tear. I was called to see her again at six, and found her in a typical eclamptic fit. During the course of the next twelve hours she had ten such fits, three of which occurred whilst she was under the influence of chloroform. After the fits had ceased the administration of the chloroform was discontinued, and one third of a grain of morphia was administered subcutaneously. She slept heavily for twelve hours, and awoke feeling fairly well, but quite unconscious of what had happened to her. Two days later she became jaundiced; bile pigments, but no bile salts, were found in the urine, and the motions were not clay-coloured,—that is, the jaundice was of the so-called hæmatogenous type. Now consider the condition of the urine.

On November 28th, the day on which the fits occurred, she passed fifty-two ounces of urine of sp. gr. 1018; this contained 1·5 per cent. of urea, and only a slight cloud of albumen. On November 29th fifty-seven ounces, sp. gr. 1017, urea 1·5 per cent., albumen distinct cloud. On November 30th fifty-two ounces, sp. gr. 1019, urea 1·6 per cent., albumen none.

No blood nor casts of any description were ever found in the urine; bile pigments were present at the time of the jaundice, but no leucin or tyrosin could be discovered. The albumen consisted chiefly of serum-albumen; only very little paraglobulin was present. Twelve days after delivery the patient left the hospital quite well, and when written to a year later stated that she had been in perfect health ever since.

I have quoted these two because they illustrate well the two commonest groups of cases which we class together under the term eclampsia. In their clinical characters they are strikingly diverse; beyond the phenomena of convulsions they possess little in common; the one is an

instance of renal disease aggravated by pregnancy, because, just as happens in a damaged heart, the affected organ has no reserve force to fall back upon when an unusual exertion is required. Every phenomenon of nephritis is there, and nothing is present which is unusual in such a case. The cause which has led to the exacerbation is removed, the acute symptoms pass off, but the severe structural changes are left as an enduring monument.

The other case has an entirely different pathology; the patient is previously in good health, the onset is sudden as a bolt out of the blue—the storm passes over, and leaves behind nothing to show its ravages. Some poisonous product has been formed or liberated; brain, liver, kidneys, and probably other organs have suffered from its effects.

The poison, in one way or another, has become excreted or rendered innocuous, and, as far as we can tell, no structural changes resulted.

I have quoted these cases in the hope of showing you that eclampsia cannot be regarded as one disease for which in every case we can adopt one definite line of treatment, and I would emphasise this further by reference to recent work by Lange.

By a series of experiments, extending over four years, he has succeeded in demonstrating that a considerable increase in the size of the thyroid gland occurs in pregnancy, and that this hypertrophy occurs most rapidly during the fifth and sixth months. We know so little about the normal functions of the thyroid, that to speculate upon the part it plays during pregnancy is almost valueless. But two facts, at present, as far as I am aware, not fully confirmed, which Lange endeavours to establish in his paper, are of great interest.

(1) That if you take two cats of equal weight, one of them pregnant, the other not pregnant, you can remove a greater mass of the thyroid of the cat which is not pregnant without giving rise to toxic symptoms, than you can do in the case of the pregnant cat. That is to say, in its economy the pregnant cat requires a greater mass of thyroid than the cat which is not pregnant.

(2) The second fact I will state as it is stated in the abstract of his paper:

"In an examination of 133 pregnant women decided enlargement of the thyroid occurred in all but twenty. In eighteen of these twenty cases albuminuria was found. Six of them developed eclampsia; in four all the premonitory symptoms, such as severe headache, dulness of vision, etc., were present, and convulsions were averted only by prompt and vigorous treatment."

Other observers, both from the experimental and from the clinical side, have arrived at the conclusion that disordered conditions of the thyroid play an important part in the production of this toxæmia. Thus Hergott * quotes an experiment, published in 1897, by Verstraeten. The thyroid of a cat was partially removed in 1892, and in 1895 this animal became pregnant. Shortly before labour commenced the cat became ill and refused to take its food; at the onset of labour coma and convulsions developed. These symptoms persisted for five days, during which time the labour made very little progress. On the fifth day intramuscular injections of a fresh extract of sheep's thyroid were administered; the improvement was almost immediate. The convulsions ceased, labour advanced, and a dead kitten was born. The cat recovered.

From the clinical point of view some interesting observations have been made by Nicholson.* In his paper he records cases in which patients have developed the premonitory symptoms, œdema, headache, loss of vision, and albuminuria, and in which these symptoms have disappeared upon the administration of thyroid extract, only to recur if the treatment were stopped.

These facts are striking and interesting, but further investigations are required before we can accept them entirely.

I have dwelt perhaps longer than I ought to have done upon the questions of pathology, because I want to make my own position clear. Briefly, I may thus recapitulate: I believe that under the term eclampsia we group together conditions which have little in common beyond epileptiform convulsions, and provisionally, I think, we may recognise three groups of cases.

(1) A condition of uræmia depending upon kidney disease.

(2) A general acute intoxication. What the toxine is and where it is manufactured we do not know. It may be (as is maintained by Müller † and by Albert ‡) of bacterial origin; it may be manufactured in the intestinal canal either by the formation of poisonous substances by a disordered digestive process or by decomposition of the products of digestion; or lastly, it may be due to disordered metabolism in the liver itself.

(3) Possibly in certain cases the poisoning may be due, as is the poisoning in cases of myxœdema and cretinism, to lack of thyroid secretion.

One other point I must draw your attention to, and that is the close connection which exists between icterus gravis and the acute toxic disease of which we have just been speaking. By icterus gravis gravidarum I mean an acute degeneration of the liver leading to rapid destruction of large areas of hepatic substance; it is indistinguishable in its clinical and pathological features from the acute yellow atrophy which occasionally occurs in women who are not pregnant and in men. I can see no essential difference between this disease and the toxic form of eclampsia,—the

* *Annales de Gyn. et d'Obstet.*, 1902.

* *Journ. of Obstet. and Gynæcol.*, July, 1902.

† *Archiv für Gyn.*, 1902, Heft 2.

‡ *Ibid.*, 1902, Heft 2.

convulsions, the jaundice, the albuminuria are present in both, and personally I am inclined to regard them as one disease, the differences which do exist probably depending upon some difference in the quantity or the properties of the toxines.

Further, since the liver is the structure most affected, other organs suffering in a lesser degree, it is possible that the poison may be conveyed by the portal circulation, and may be formed in the intestinal canal; or it may be that the liver is at fault, and fails to deal in its proper manner with some product of digestion which is brought to it. The latter theory has experimental evidence to support it, since convulsive fits often follow the making of Eck's fistulas. Having thus indicated the lines upon which I think this discussion should be conducted, it remains only for me to briefly introduce it, and to leave its elaboration to subsequent speakers.

With regard to treatment, two extreme views are met with to-day. One school of obstetricians advocate Porro-Cæsarean section. Another school recommends that the women be left entirely alone, that labour should not be induced, and that drugs should not be administered; and between these two extremes every shade of opinion is found. I will indicate one or two questions which I think we may with advantage discuss to-night; we should, I think, have clear views upon these points, because any one of us may at any time be called upon to treat a case of eclampsia. And the first question I will raise is this: "If eclampsia threatens or is imminent should labour be induced?" and "supposing labour to have commenced, how far should we by artificial means hasten its completion?"

Dr. Willett has kindly consented to speak upon these points, so I shall content myself with very briefly stating my own views without entering into any details. In cases of uræmia, where the symptoms depend upon renal inadequacy, it is important to relieve the kidneys of the additional work which has been forced upon them. So long as the child remains in the uterus the kidneys of the mother have to perform excretory functions for two organisms; by emptying the uterus we diminish the demands which are made upon them; therefore in cases of uræmia I believe we ought to bring about delivery as rapidly as *is perfectly consistent with the safety of the mother;* I would not use accouchement forcée, and I would not use Borsi's dilator.

In cases of toxic eclampsia, however, there is evidence to show that there is not the same urgency in inducing labour; we know that very often, as in a case I have quoted, the fits do not commence until after delivery; we know that very often when they appear before or during labour they persist after its completion; and further, that in many cases there is no evidence of diminished excretory activity. For these reasons I believe that to hasten delivery is not by any means an essential part of the treatment.

The second point I would suggest for consideration is, "What treatment shall we adopt during the convulsive fits?" This topic Mr. Hunt will introduce, and therefore, for the present, I entirely omit it.

But if I may be allowed to occupy your valuable time for a few minutes longer, I would like to direct your attention to the no less important question of the general treatment of the condition.

Let us in the first place ask ourselves, "Can we do anything to stave off an attack of eclampsia? Is there a preventative treatment?"

First let us take those cases to which I would apply the term uræmia rather than eclampsia.

I say, without hesitation, that the majority of these cases ought not to reach the stage of uræmia. The warnings are, as a rule, many and distinct. Albuminuria in a pregnant woman is always to be considered seriously, and it is surprising how often one finds that medical practitioners of experience fail to realise the gravity of the symptom. Every case of albuminuria, then, requires treatment; and if in addition such symptoms as œdema, headache, loss of vision, supervene, the gravity of the position to the patient can hardly be over-estimated; but the recognition of the early phases of the disease is an all-important matter. These conditions, then, should be carefully treated: absolute rest in bed, most careful dieting, and every precaution taken to maintain the excretory activity of the kidneys; and should these measures not prove sufficient to establish a marked improvement, the uterus must be emptied. In these cases, then, uræmia is often a preventable disease.

Turning, then, to a second group—a group at present very little recognised in England, and whose existence can hardly yet be said to be proved,—the cases of thyroid inadequacy. Lange claims to have averted the fits in many cases by the use of thyroid extract.

The third group of cases—the instances of acute toxæmia—present greater difficulties; the onset may be of extreme suddenness, and the warnings very few. Further, we know neither what the toxine is nor where it is formed.

In these acute and sudden cases, then, until we know something more of the lesion, there are few, if any, measures we can apply. But once the threatenings of the disease have become manifest, every care and every precaution must be taken. We must bear in mind two points; (1) that we are dealing with a toxæmia, and (2) that the convulsive fits are often excited by external stimuli.

The fits are dangerous to the life of both the mother and the child, and it is our duty to do everything we can to ward them off. The patient must be confined absolutely to her bed in the quietest room available. The room should be kept darkened; beyond the doctor and the nurse, no one should be allowed in it, or if it can possibly be avoided even to pass the door. The patient must be fed, but only with liquid diet. All forms of examination

should be dispensed with to as great a degree as possible. It will be necessary to make one satisfactory examination, and also to obtain a catheter specimen of the urine. I would recommend that chloroform be administered before these things are done. These women take chloroform well, and I have never seen any harm result from its administration.

Should muscular twitchings supervene—even though no fits occur—sedatives should be administered, and to my mind there is no drug which compares in this respect with morphia. It should be administered hypodermically—for patients with eclampsia are very apt to vomit, and under these circumstances one can never be sure what quantity of the drug has been retained. Other drugs of value are chloral and bromide; they must be given in large doses, and it is often advantageous to administer them by the rectum.

What can we do to combat the toxæmia?

In the first place we can promote excretion by the bowels, by the kidneys, and by the skin. I have already said that there is some reason in the suggestion of an intestinal poison, and therefore the bowels should be made to act freely,—calomel, saline purgatives, and in urgent cases croton oil should be administered. To my mind the exact drug one uses is not a matter of great importance provided the desired result be obtained.

The excretion of the skin may be encouraged, but I regard this as a very secondary matter; it may be a matter of very great importance in uræmia, but in eclampsia to employ such violent methods as hot air or vapour baths is in my opinion not the best treatment.

There is no evidence whatever to prove that the poison is excreted by the skin, and I have myself seen the fits directly provoked, as I believe, by the hot air bath; at any rate, the fits recommenced as soon as we put the patient in the bath, and stopped as soon as we took it away again.

There is another method of increasing the secretion of the skin, of which I speak only to warn you against it. I refer to the injection of pilocarpine. There is perhaps no method of treatment more dangerous than this; it increases the cutaneous secretion undoubtedly, but it stimulates in a precisely similar fashion the bronchial mucous membrane. The bases of the lungs, after the fits, are often in a condition of intense congestion and of œdema, and to increase the secretions of the bronchial mucosa under these circumstances is often to sign the death-warrant.

With regard to the kidney, undoubtedly free excretion of urine is an important matter, but I have shown you in the case I have quoted that eclamptic convulsions may occur in cases in which the normal quantity of urine is being passed. Furthermore, the experiments of Bouchard by which he claimed to prove that the toxicity of the urine was diminished during the time of the fits, and increased afterwards, have been shown to be unsatisfactory, for he took no pains to keep the urine sterile, and paid no attention to the temperature at which it was injected, and other workers who have corrected these errors have failed to confirm his results.

We have, then, no positive proof that the poison of eclampsia is eliminated by the kidneys, but I regard it as extremely probable that they do play an important part in the process, and therefore I urge that every means be taken to promote kidney secretion. We have various means in our power of doing this. In the first place we may use drugs; and there is one drug, not found in the British Pharmacopœia, which is of great value, and that is old unsweetened gin. In several cases both in uræmia and in eclampsia I have used this remedy, and I believe it to be superior to most other diuretic drugs. I said just now that we had no proof positive that the poison was excreted by the kidneys, but it must be remembered that a very considerable number of these acute cases are accompanied by uræmia or by partial suppression, and that when the flow of urine is re-established the symptoms subside. It may be, of course, that the re-establishment of the flow of urine is merely an indication of the improvement in the condition; or, on the other hand, it may be that free secretion of urine is the cause of the improvement, we cannot say; but in the present state of our knowledge the indication for treatment directed to this end is a clear one, although some facts are hard to explain. How is it, for instance, that in death from calculus anuria or from the removal of an only kidney convulsive fits do not occur? Rose-Bradford's recent work has gone far to demonstrate that in uræmia the convulsive fits depend, not upon the presence in the blood of bodies which should normally be excreted by the kidneys, but upon a special poison, or upon the lack of some internal secretion which it is the function of the kidney to provide. How far may this work be made to apply to eclampsia? We do not know, but its results may be much more far-reaching than is dreamed of at present.

But to return to our question: is there any other mode of producing diuresis? There is, and it is by injection of large quantities of saline fluid.

This method of treatment merits a little further notice. One of the quickest ways in which we can establish diuresis in a healthy animal is by the injection, either subcutaneous or intra-venous, of large quantities of fluid, and within the last few years this method has been somewhat extensively used in the treatment of these cases of eclampsia. In a healthy animal diuresis is established by this means almost instantaneously; in eclampsia its establishment is a much slower matter. In some of the recorded cases the interval has been so long that one wonders whether the improvement is not *post* rather than *propter*.

The improvement which often follows this method of treatment has been accounted for in somewhat different ways. According to some observers, the coagulability of the blood is greatly increased in eclampsia, and some

would even attribute the condition to small cerebral thrombi and emboli, and, according to the people who adopt this view, the action of the fluid is to dilute the blood and render it less coagulable; whilst others, and I think with much more reason, believe that the only action of the fluid is to increase the flow of urine.

I regard this method of treatment as, in many cases, a valuable one, and I believe its chief action is simply to increase the flow of urine.

I have already trespassed too long upon your time, and therefore I shall leave to those who follow me such questions as the advisability and indications for bleeding (often a very valuable aid to treatment); the administration of chloroform, which in my opinion should be given to a greater or less degree during the whole of the second stage; and the treatment of the convalescence.

I apologise for the crude fashion in which I have put these matters before you; they are matters of the very first importance, but whilst we are still so much in the dark with regard to pathology, our treatment must, to a great extent, be empirical.

I think we should only be wasting time were we to dwell upon old theories and old forms of treatment which have now practically been abandoned; many of these have played an important part in the evolution of our modern ideas, and through them our pathology has risen on stepping-stones of her dead self to a truer position.

I will ask you to remember that this evening we have met for a discussion, and I hope sincerely that most of those present will contribute.

Abernethian Society.

AT the meeting of the Abernethian Society held on February 26th Dr. Williamson read a paper on "The Treatment of Eclampsia," which appears elsewhere.

In the discussion that ensued Dr. J. A. Willett said: Mr. President, I think our best thanks are due to Dr. Williamson for the very able way in which he has summarised our knowledge of the causation and pathology of eclampsia. The views he has expressed as to the *rôle* played by the thyroid are very interesting, but I do not think that they will help us very much in treatment, for it is very difficult to definitely diagnose a slight diminution in the size of the thyroid; and unless there are some special symptoms in those cases of eclampsia due to thyroid inadequacy, I fail to see how we are to know when to give thyroid extract with advantage.

Taking the subject of the treatment of eclampsia occurring during pregnancy, I assume that the patient has the usual symptoms—headache, vomiting, ocular symptoms, and œdema, but has not had as yet a convulsion. I quite agree with Dr. Williamson that we must try and determine to which of the two groups the symptoms are due. In either case it would be right, I think, to try the effect of palliative treatment. In those cases due to renal inadequacy, unless the symptoms rapidly improve in a very short while I do not think that the treatment should be persisted with, for we have to remember (1) that the disease of the kidney is incurable and progressive; (2) that the symptoms are due to the increased work thrown upon them by the products of the portal metabolism,—or in other words, the child being the exciting cause of the disease, the indication would be to remove such cause; and (3) if, as so often happens, the child dies, the symptoms do not necessarily diminish, nor is the risk of eclampsia abolished, so there is no ground for temporising, in the hope that if the child dies the symptoms will cease. At least this is the experience I have had in one or two cases, and I should like to ask Dr. Williamson his views,—whether, if the child dies, the albumen disappears from the urine and the other symptoms abate. As regards the toxæmic class, I think that as a rule palliative treatment is all that is wanted, for as the kidneys are not permanently damaged, but their functions more or less paralysed by the severity of the poison, it seems to me that if we can eliminate the poison by appropriate treatment the symptoms will cease. Also if our treatment fails, at least we have the satisfaction of knowing that no permanent harm has been done. But it often happens that there are no warnings, and the first symptom of the disease is a convulsion, as in a case at Queen Charlotte's Hospital when I was resident there. I believe in such cases that the right thing to do is to induce labour in the interests of the child, provided of course that it is viable, for I feel pretty sure that the longer it is left *in utero* the more likely it is to be born dead. Supposing the patient to be in labour when the first fit occurs, we must be guided by the severity of the case. The two symptoms which I believe to be the more important are (1) the quantity of urine secreted, which I think of greater prognostic value than the amount of albumen and urea it contains; and (2) the condition of the patient between the fits, the return of consciousness indicating a hopeful outlook, coma the reverse. If the patient is passing a fair quantity of urine, and recovers consciousness between the convulsions, I would give a hypodermic injection of morphia and leave her alone until the commencement of the second stage, when forceps may be applied in the interests of the child.

In the graver cases I am sure the indication is to deliver as quickly as possible, provided that the method employed does not in any way entail an increased risk to the mother's life; and whatever method is employed, it should be one which can be used as well in private as in hospital.

Various means have been suggested or employed, but I will only refer to three.

1. *Cæsarean section.*—Objections to this mode of delivery, it seems to me, are obvious; its only recommendation is its rapidity. Skilled assistance is immediately necessary. The surroundings may be unfavourable, which will add a great risk of sepsis to the other dangers of the operation; the general condition of the patient is most unsuitable for an abdominal section; and lastly, a patient who is so bad as to require any operation of this kind is hardly likely to survive it.

2. *Bossi's dilator.*—In skilled hands this instrument seems successful, as the results of the cases reported by Dr. Ballantyne prove. It is said to dilate the os fully in thirty minutes; but any rigid instrument worked by a screw may prove extremely dangerous from tearing instead of dilating, especially if the dilatation is conducted at this pace.

3. *C. de Ribes' bag* is, I think, the most useful method. The os can be safely and rapidly dilated. The only risk is that of sepsis, which is known, and can be guarded against. In using the bag the membranes should be ruptured, and some liquor allowed to escape. In this way the risk of over-distension of the uterus, and consequent weak pains, is avoided, as well as that of causing a mal-presentation. If, after filling the bag, steady traction is maintained, it will usually be expelled in under an hour.

It must be admitted that such manifestations tend to increase the number of convulsions, and for this reason $CHCl_3$ should certainly be administered during their performance. There is no evidence, to my mind, that the presence of the bag *in utero* excites convulsions; from what I have seen I think the convulsions depend much more on the rapidity of sequence and severity of the pains.

In conclusion I should like to ask Dr. Williamson and subsequent speakers their opinion of the risk of the condition relapsing in future pregnancies. I have only seen one case who had eclampsia of the toxæmic variety in her first pregnancy. The second confinement took place without any untoward symptom.

Mr. Hunt said: Mr. Williamson has kindly asked me to discuss chiefly the treatment of the actual fits themselves. It may be well to begin by asking one's self the question, "What evils may result if the fits are permitted to go on unchecked?" The woman, firstly, will certainly tend to exhaust herself, and may bite her tongue or otherwise injure herself. Secondly, she may actually die in a fit. There is frequently some œdema of the glottis, and a slight spasm occurring during a fit may cause death. Another and rarer cause of death is hyperpyrexia, which will occur only if the fits are violent and prolonged. I have known of a case where the temperature was 106° when the medical attendant arrived at the house, and Dakin quotes cases of temperatures up to 107° and 108°. This high temperature seems to be similar to that which occurs in tetanus, in

which disease I have seen the temperature rise rapidly during a violent spasm to 111° F., and the patient die apparently from this cause. Thirdly, the child may die during a fit. Thus in treating the fits one must be prepared to protect the tongue, to open the jaw with a gag and pull the tongue forward, or even in some cases to perform tracheotomy. To restrain the fits themselves chloroform is without doubt the most useful agent, and its administration should be continued for as long as is necessary. It lessens the cyanosis, and does away with the risk of spasm of the jaw, etc. It lessens the exhaustion, and entirely prevents any hyperpyrexia from prolonged muscular contraction. Lastly, it renders all vaginal manipulations easy. Morphia is another drug which is extremely useful. It should, of course, always be administered hypodermically, and it seems to be usually advisable to give half a grain at once, and then repeat in smaller doses to the extent of keeping the pupils contracted. According to Mr. Williamson's valuable suggestion as to the two kinds of eclampsia, it would seem more advisable to administer the morphia in the "toxic" form than in the uræmic form. Other drugs, such as chloral and bromides, are of subsidiary importance. Most authorities are now agreed that pilocarpine should never be given, since its use is very often followed by an increase in the already considerable œdema of the lungs.

Several other members took part, among whom were Messrs. Worthington, Sale, Elmslie, and Colt.

Dr. Williamson then replied to many questions that were put, and the meeting was adjourned.

Amalgamated Clubs.

RUGBY FOOTBALL CLUB.

"A" TEAM.

ST. BART.'S "A" *v.* SURBITON "A."

Played at Winchmore Hill on Saturday, February 14th, and resulted in a very even game, the final score being a try each. Brewer scored the Bart.'s try. The forwards in this game showed up very well. Team:

A. J. Kendrew (back); N. M. Wilson, E. Harrison, B. A. Keats, F. H. W. Brewer (three-quarters); W. R. Collingridge, R. S. Townsend (halves); — Hoskyn, A. J. Symes, R. Jamison, — Arnold, S. Trevor-Davies, A. R. Snowdon, — Fuller, E. R. Jones (forwards).

ST. BART.'S "A" *v.* ST. THOMAS'S "A."

Played at Winchmore Hill, February 22nd, and resulted in a win for Bart.'s by 1 goal 2 tries to 1 try. It was an excellent game, and the outsides handled well in spite of the greasy ball. Tries by Harrison (2) and Wilson (1). Team:

C. H. Cross (back); N. M. Wilson, P. R. Parkinson, E. Harrison, B. A. Keats (three-quarters); R. S. Townsend, F. H. W. Brewer (halves); R. M. Millar, A. J. Symes, A. R. Snowdon, F. Trewby, G. W. Lloyd, H. Wenham, E. R. Jones, — Haman (forwards).

ST. BART.'S *v.* ST. GEORGE'S.

1ST ROUND.

This match was played at Richmond under very favourable weather, the ground being perfect. Bart.'s won the toss, and elected to play with the wind. George's kicked off, and Owen returned into touch. Play was of a very scrambling nature, and within twelve minutes of the start the Bart.'s forwards rushing the ball to the opponents' goal-line, Morris succeeded in scoring between the posts. Lee's kick being charged down, a fast rush by George's finished with Bailey very nearly scoring, Bart.'s having to touch down. Just before half-time a combined movement of the Bart.'s three-quarters enabled Lee to score, though he was unable to convert.

In the second half very little of importance happened, the play being principally confined to the forwards, and in spite of Bart.'s being in the George's "25" nearly all the time, they were unable to score.

Result: St. Bart.'s 2 tries, St. George's *nil*.

In St. Bart.'s there was plenty of good individual play among the forwards, but no combination; also the centres played a good game. In St. George's Bailey played excellently. Team:

E. S. Marshall (back); E. Harrison, C. S. Lee, H. B. Owen, W. H. Thompson (three-quarters); W. H. Hamilton, W. G. Loughborough (halves); H. T. Wilson (capt.), A. R. Neligan, J. Morris, R. I. Douglas, R. M. Rankin, R. Hoskyn, H. MacDonagh, A. J. Symes (forwards).

ST. BART.'S *v.* GUY'S.

2ND ROUND.

Played at Richmond on Tuesday, January 25th. Wilson won the toss, and decided to play with the wind. Guy's kicked off, and after some loose play in Bart.'s "25" Morgan obtained the ball, and transferring to Alcock, the latter scored three minutes after the start. After this Bart.'s in a series of scrums took the ball into Guy's "25," where some close play took place forward. One three-quarter getting the ball, a chance of scoring was missed by the wing not taking his pass. Guy's then constantly getting possession, and by brilliant outside play, Morgan scored twice, both being converted. After this the ball was again carried into the Guy's "25," where good combined rushes on the part of our forwards resulted in Guy's having to touch down. Soon after half-time was called. All through the second half, in spite of our forwards fully holding their own, we could not prevent the Guy's three-quarters from scoring, tries being obtained by Alcock, Orpen, and Saunders in succession, Alcock's try being converted by O'Brien. Shortly before time Marshall, following up his kick, charged down Harrison's return; this enabled Bart.'s to press hard, and Miller got over far out, Lee narrowly missing a difficult kick.

Result: Guy's 3 goals 3 tries, St. Bart.'s 1 try. Team:

E. S. Marshall (back); G. D. Drury, C. S. Lee, H. B. Owen, P. R. Parkinson (three-quarters); W. H. Hamilton, W. J. Loughborough (halves); H. T. Wilson (capt.); A. R. Neligan, J. Morris, R. I. Douglas, R. M. Rankin, R. Hoskyn, R. M. Miller, G. H. Dive (forwards).

HOCKEY CLUB.

INTER-HOSPITAL HOCKEY COMPETITION.

ST. BART.'S *v.* GUY'S.

At Blackheath on Thursday, February 5th. The game opened with some scrambling play round the touch-line, about mid-field. Then Gray gave "sticks," and transferred play to our circle. But after some scrambling play the forwards, headed by Glenny, took the ball right down the field, where Glenny scored after a fine run. Play, however, soon came back, and Furber saved well. Guy's here were having every bit as much of the game as we were. Raikes then tried hard to get the ball away from touch, but was unsuccessful, and play waxed fast and furious round our goal, but Hill saved cleverly. Once more Raikes was very prominent, but Guy's came back to the fight, where Coalbank cleared well. We then suffered many anxious moments; there was a fine rush by the Guy's forwards, but one of their men by giving "sticks" gave us relief. Bart.'s then took the ball right up the field, only to be repulsed, when "sticks" once more saved us. Then play once more settled down in our "25," after some scrambling play in Guy's half, but Whitby played a fine game throughout. Coalbank and Page help us materially. But "sticks" against us once more brought Guy's to the attack, but Raikes, Glenny, and Barton took the ball away, but Barton unfortunately hit too hard, with the result of a bully on their "25." Then Guy's made a splendid rush and scored goal all. This put us on our mettle, and from a forward rush Griffin just missed scoring. Next Barton was again, as always, prominent in stopping a Guy's rush. Once more back we went to the Guy's "25," and Raikes by getting "off side" missed what looked to be a certain score. We then kept up the pressure, but Haines and Gray both missed chances; however, presently Raikes centred finely, and Haines shot a beautiful goal. Soon after the re-start Gray got "off side," and Guy's had a chance but missed the goal. However, Guy's kept up the pressure, and twice Whitby saved magnificently. Then twice more "sticks" saved us; then there came some scrambling play in mid-field and half-time came, leaving us leading by 2 to 1. At the re-start Guy's looked dangerous, but Whitby saved well. Then a Guy's man got "off side," but Furber in taking the hit gave "sticks" and nearly let them in, but we once more got clear, only to be repulsed with a corner given against us followed by a goal. Soon after this Bart.'s made a fine dash, headed by Haines and Glenny, and Griffin scored from a scramble round their goal, in which Raikes was distinctly prominent. Then Haines made a good run, and "sticks" being given against Guy's, Griffin added one more to our score. Guy's came back to the attack, and Whitby was once more called upon, to which he responded manfully. Then Glenny got away, but just as things looked hopeful Strange of Guy's got hit in the eye, and play was stopped for a few minutes. Then Guy's made a fine rush followed by a counter rush by Bart.'s, in which

Glenny scored from a good centre by Raikes. Twice more in succession Whitby was called upon to save, but Bart.'s got clear by a good rush, in which Page was prominent, only to be driven back by a counter rush by Guy's, but their shot went wide. Once more we got back to mid-field, but Guy's rushed us back once more, and Barton was again prominent by stopping them. Then came another anxious moment, but Whitby and Page cleared well. Then the whole team seemed to combine in a grand rush, from which Glenny scored. But we were once more driven back, this time to be relieved by our captain, who worked magnificently throughout and led his men well. Then Coalbank, Whitby, and Barton were each in turn prominent; then once more down we went to the Guy's goal, only to be driven back by Haines getting "off side." And soon after, after some scrambling play in mid-field, the whistle sounded its welcome and victorious note, leaving us the winners of a very fast game by 6 goals to 2. The whole team worked hard to a man, but Glenny, Barton, Whitby, and Hill were especially prominent. Team:

F. Whitby (goal); M. R. Coalbank, L. G. H. Furber (backs); H. B. Hill, B. H. Barton, G. F. Page (halves); H. Gray, C. T. Raikes, W. B. Griffin, E. T. Glenny, R. L. Haines (forwards).

A few noble spectators braved the wintry wind to cheer us on. We hope to see more in the next match.

ST. BART.'S *v.* LONDON.

Played on Thursday, February 26th, at Blackheath. The game started with a bit of scrambling play in the centre. Then London made a rush up to our goal, but were driven back, and for some while play was of a very even character; but after a while our forwards, playing well together, especially Haines and Glenny, transferred play to the London circle, but then, after being driven back to the halfway, we got really going, and notched the first point through Glenny. Then for some time we still besieged their circle, but without avail. However, London came back to the attack and got a corner, but Raikes, getting the ball, with a fine run went the length of the field, and notched our second point. Soon after the re-bully off the London forwards made an effort, and Gibson, using his pace to the best advantage, scored for them. This did us good, as it made us stick to it, and eventually from a scramble round their goal Griffin scored; and soon after half-time was called, leaving us leading by 3 goals to 1. After half-time London made one dash to start with, and for a few minutes play waxed hot and furious round our goal, but our defence, materially assisted by Page's fine play, proved impermeable. And then we had things much our own way, with fast and occasional but rather faint-hearted rush by London. We scored again through Glenny twice, the first after a pretty run, and Griffin once. Griffin and Raikes did a lot of very useful work, but showed no gallery play. Glenny shone conspicuously the whole game, as did also Page and Barton, the former sticking to Gibson, certainly one of London's most dangerous forwards, like a leech. The whole team, considering they had only played together once before, showed quite a nice amount of combination, and every one played up hard, even though never really extended. Whitby in goal had little to do, but did what he had very usefully. The team were excellently led by their hard-working captain, H. B. Hill.

Bart.'s now meets the winner of Thomas's and University, probably Thomas's, in the final, and we wish them the best of luck. Team:

F. Whitby (goal); L. G. H. Furber and L. L. Phillips (backs); H. B. Hill, B. H. Barton, G. F. Page (halves); H. Gray, C. T. Raikes, W. B. Griffin, E. T. Glenny, and R. L. Haines (forwards).

THE UNITED HOSPITALS HARE AND HOUNDS.

The efforts made to reorganise this club have not been altogether successful. Last year Bart.'s had a walk-over for the Inter-hospital Challenge Cup, no other hospital being able to get together a full team. This year it fell to Guy's to do the same, as we were not able to raise the necessary team of three men, although G. W. Lloyd and W. H. Barnett moved out, the former getting third place, while the latter ran quite untrained.

J. G. Gibb, who could have won the race easily had he run, was unable to, owing to having a lecture on the same afternoon. There does, indeed, seem to be a change come over Bart.'s men when they cannot sacrifice one lecture to represent their Hospital in an inter-hospital competition. The same applies to W. H. Orton, who had been running all the season for the club, and A. C. Wilson, neither finding it convenient to turn out for the race.

The team which won the Cup last year was formed by F. S. Lister, J. G. Gibb, and P. Gosse. Lister and Gosse were both "crocked," and so unable to turn out in this year's race.

The first race of the season was a five-mile handicap, which was won by G. A. Simmons, of St. Thomas's.

The first team race was against the South London Harriers, who were defeated by 11 points.

The next race was against Wellington College; but on this occasion Gibb was running for the L.A.C. against Cambridge, and O. S. Norton (Guy's) for the Blackheath Harriers against Oxford. The result was a defeat by 2 points.

The race against the Blackheath Harriers was run on December 6th, when the ground was frozen so hard that in crossing the first bit of plough both J. G. Gibb and P. Gosse broke down with sprained ankles. The result was a win for the Bart.'s Hospital, although the United Hospitals Hare and Hounds supplied the first man home in Norton.

The races against the Thames Hare and Hounds and the Ranelagh Harriers had to be abandoned owing to the hard state of the ground, and Oxford University Hare and Hounds scratched, as they could not raise a team so late in the season.

In the race against Cambridge University Hare and Hounds the Hospitals were represented by Messrs. Gibb, Lloyd, Norton, Carr, and Simmons. The race was over a course of seven and a half miles with fairly heavy going. The result was a win for Cambridge by 9 points. The first man home was A. R. Welch, who beat Gibb by a yard or two.

The Inter-hospital race was won by L. Pilbeam, the best of the Guy's Hospital runners, quite easily, beating T. E. Carr of the same hospital by three minutes. Had other Bart.'s men the same patriotic feeling towards their Hospital as G. W. Lloyd and W. H. Barnett showed, no doubt the Cup would now be an ornament to the Library for another year. It is hoped that the race will in future be run before Christmas, when it is easier to get together a team.

Examinations.

M.D. Cambridge.—A. M. Ware.

UNIVERSITY OF LONDON.

Intermediate M.B. Examination; entire Examination—*1st Division.*—C. Clarke, G. S. Morse. *2nd Division.*—H. D. Clementi-Smith, C. H. Cross, N. Bennett-Powell.

Excluding Physiology.—A. Barber.

Appointments.

BURROWS, HAROLD, M.B.(Lond.), F.R.C.S.(Eng.), appointed Assistant Surgeon to the Bolingbroke Hospital.

* * *

STONE, G. W., M.R.C.S., L.R.C.P., appointed Surgeon to the s.s. "Johannesburg."

New Addresses.

ADAMS, P. E., Hartley Wintney, Winchfield, Hants.

JAMESON, R. W., Plague Office, Point, Durban.

PICKETT, J., Vale Towers, Tunbridge Wells.

STANLEY, H., 43, Pevensey Road, St. Leonards-on-Sea.

VAUGHAN, A. LL., St. Nicholas Street, Diss, Norfolk.

WINKFIELD, C. F., Belmont, Whetstone, Middlesex.

Marriage.

POWELL—BRENTNALL.—On February 24th, at St. Matthew's Church, Upper Clapton, by the Rev. J. Cullin, M.A., Vicar, Josiah Cecil Powell, M.R.C.S., L.R.C.P.(Lond.), youngest son of Henry Penk Powell, of Stoke-on-Trent, to Adeline Marie, eldest daughter of Charles Brentnall, 131, Clapton Common, N.E.